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TREATMENT OF LUPUS ERYTHEMATOSUS WITH MEPACRINE.

Results and Relapses during a long Observation.

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In the following article, the term "lupus erythematosus" (L.E.) refers only to the fixed type of lesion and, unless specifically mentioned, does not include the disseminated forms.

As the ætiology of L.E. is unknown, rational therapy in the strict sense of the word is not feasible. Our possibilities of treatment rest exclusively upon clinical experience, and as none of the remedies hitherto employed has been ideal, the new drugs available in the treatment of this disease have been the subject of much interest.

In recent years, attention has been focused mainly on mepacrine (atabrine, quinacrine hydrochloride) and chloroquine. The encouraging results with these drugs reported by others prompted us to give them a trial, and we have now used them for more than two years—mepacrine throughout 1953 and the greater part of 1954, and chloroquine since July 1954. A report of our results with mepacrine is presented in the following. We attached importance to having a fairly large number of patients and aimed at a long period of observation after the termination of therapy. It is too early yet to evaluate the results of chloroquine therapy.

The Effect of Mepacrine on Lupus Erythematosus.

Prokopchuk (1940) appears to have been the first to use mepacrine in the treatment of lupus erythematosus. Sorinson (1941) adopted the form of treatment advocated by Prokopchuk, but otherwise interest in this use of mepacrine was not revived until Page published his study in 1951.

Prokopchuk treated 35 patients with quinacrine hydrochloride 100 mg. *t.i.d.* for ten days followed by an interval of 10–11 days, whereupon the treatment was resumed. Page (1951) treated 18 cases of lupus erythematosus with mepacrine in doses varying from 100 to 300 mg. daily. 9 patients showed an excellent result, 6 good, 3 slight, and one nil. The duration of treatment was up to two months.

Since then the principles of mepacrine therapy have been fairly uniform : 200–300 mg. daily until yellow pigmentation becomes marked, and then a lower dosage of about 100 mg. for some length of time.

The results published by a number of investigators are summarized in Table I. As the various authors have not used the same classification of cases, the table should be taken only as an expresssion of our interpretation of the available data.

TABLE I.—*Lupus Erythematosus Treated with Mepacrine (Atabrine).*

	Number of cases.	A Excellent.	B Improved.	C None or doubtful effect.	Positive results per cent. (A+B).
Prokopchuk, 1940	35	?	?	?	—
Sorinson, 1941	51	12	17	22	57
Page, 1951	18	9	6	3	83
Sommerville *et al.*, 1952	23	4	15	4	83
Cramer and Lewis, 1952	6	5?	—	1	83
Wells, 1952	12	3	6	3	75
Sawicky *et al.*, 1952	30	6	15	9	70
Black, 1953	60	10	23	27	55
Kaminsky and Knallinsky, 1953	61	10	38	13	79
Harvey and Cochrane, 1953	62	23	14	25	60
Kierland *et al.*, 1953	52	17	24	11	79
Rogers and Finn, 1954	45	17	7	21	53
Helanen, 1954	36	10	21	5	86
Total	456	126	186	144	68
Present series	97	31	39	27	72

MATERIAL.

In 1953 and 1954, 99 cases of L.E. were treated with mepacrine. Only one case was of the subacute, disseminated form ; in all the others the lesions were of either the discoid or the purely erythematous type. In one case treatment had to be stopped after two days on account of dyspepsia, so that this case is included only in the discussion of side-effects. The following report deals with the remaining 97 patients.

When we began to use mepacrine therapy we thought it desirable to admit the patients to hospital at the start of treatment, as this would offer the best possibility of counteracting any side-effects. Later in our study we adopted out-patient treatment. As it is a common experience that a stay in hospital has in itself a beneficial effect on lupus erythematosus, we hereby introduced a

source of error in our evaluation of therapeutic results. To obtain clarification on this point, the patients were divided into two groups, those treated while in hospital, and those treated exclusively as out-patients. The period spent in hospital varied from 2 to 14 weeks, average 4·5 weeks.

As we could not know beforehand whether the two groups of patients (in-patients and out-patients) were equivalent, partly because most of the hospital patients started treatment in 1953 and most of the out-patients in 1954, we

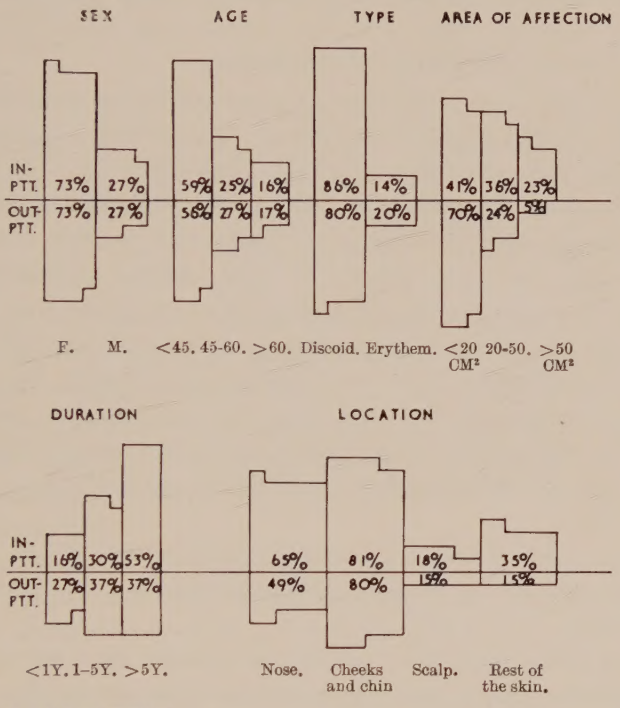

Fig. 1.—Distribution of the Material.

subjected the two groups to a careful analysis. Fig. 1 shows the numbers of patients treated and their percentage distribution within the two groups. The patients were classified according to sex and age, the type, site and extent of their lesions, and the duration of the disease.

56 patients were treated while in hospital, 41 as out-patients. From Fig. 1 it will be seen that the two groups did not differ in regard to sex and age distribution. Lesions of the erythematous type were seen somewhat more frequently in the out-patients than in the in-patients, whereas the lesions were more extensive and the duration of disease longer in the in-patients than in the out-patients. As regards the site of lesions there was practically no difference between the two

groups. As the differences were so slight it was deemed justifiable to make a direct comparison of the two groups of patients.

Sex Distribution.

There was a great preponderance of women over men (about 3 to 1); this ratio corresponds to what is usually found in this disease. Analysis showed no influence of sex upon the type and extent of lesions, and the duration of disease, but plaques on the scalp seemed to be somewhat more frequent in the women.

Principles of Therapy and Period of Observation.

As mentioned previously, 56 patients started treatment while in hospital, and 41 were treated exclusively as out-patients. Most of the in-patients continued therapy for some time after they had been discharged.

The doses given were 2 tablets of 100 mg. daily during the first eight days, then 300 mg. daily until yellow pigmentation became marked, and subsequently 100–200 mg. daily for some length of time. Yellow pigmentation usually occurred about 3 weeks after starting mepacrine. In a few cases, side-effects—particularly dyspepsia—made it necessary to suspend treatment before pigmentation became marked. The total dose ranged between 1·6 g. and 49·8 g., and the most common total dose was 20 g. In the in-patients the average dose was 21 g., in the out-patients 14 g., and in the total material 18 g. An approximation of the period of treatment is obtained by calculating 1 g. as equal to 1 week (Table IV).

The period of observation is calculated from the end of the first course of mepacrine therapy. It was between 3 months and 21 months, and the average was 9·7 months.

There was a fairly even distribution throughout the year of the dates of starting treatment, with a slight preponderance in the months of April, May and June, in 1953 as well as in 1954.

Results.

The results of therapy were assessed at the end of the first course of mepacrine therapy. In a number of patients treatment was later resumed either with mepacrine or, since July 1954, with chloroquine.

Evaluation of the results of treatment of L.E. presents some difficulty as it is exceptional to obtain freedom from symptoms. The problem is twofold: (i) to provide fairly well-defined criteria of the degree of improvement and (ii) to secure that individual cases are evaluated in a uniform manner. To solve these problems we worked out a table of evaluation in which the following factors were taken into consideration: (i) erythema, (ii) infiltration, (iii) circumscription, and (iv) scaling and follicular keratosis. Each of these factors was evaluated

separately and classified as follows: disappeared (disapp.), reduced (red.) or unaltered (unalt.). On the basis of these individual evaluations, the results of therapy were divided into three groups in accordance with the following table (to be read vertically):

I. *Excellent Result.*

Erythema	Disapp.	Disapp.	Disapp.	Disapp.
Infiltration	„	„	„	Red.
Circumscription	„	„	Red.	Disapp.
Scaling and follicular keratosis	„	Red.	Disapp.	„

II. *Improvement.*

Erythema	Disapp.	Disapp.	Red.	Disapp.
Infiltration	„	Red.	„	Red.
Circumscription	Red.	„	„	Unalt.
Scaling and follicular keratosis	„	„	„	Red.

III. *Result Nil or Questionable.*

Comprises all combinations not previously mentioned.

To aid in the evaluation, all patients were photographed both before and after the course of treatment, and all were evaluated by the same investigator.

It was made a prerequisite for classification of a case within Group I or II that all the plaques fulfilled the criteria mentioned. Thus, if a few plaques cleared during the course of treatment while others remained unaltered, the case would fall within Group III.

It is our impression that this procedure contributes essentially to solve the problem of a uniform and well-defined evaluation of the therapeutic results. In any case, the method here employed is sure to be better than a rough clinical estimate.

Of the 97 patients treated, 31 (32%) showed an excellent result (Group I), 39 (40%) improvement (Group II), and 27 (28%) a result that was nil or questionable (Group III). In other words, a total of 70 patients (72%) responded favourably, and 27 (28%) did not.

In view of the comparatively small numbers involved we thought it desirable to calculate the statistical error of the results. It will be appreciated that a statistical test will correct only those errors which arise because the numbers are comparatively small, and that errors of a systematic nature, for instance uncertainty as to the category within which the result should be classified, are not corrigible by statistical methods. The calculations below are based on the following formula: $\delta = \sqrt{N \times p \times q}$, where δ is 1 standard deviation, N the number included in the test, p the probability of the result in question, q the probability of the remainder. We chose to give the result with the limit of 2 standard deviations which corresponds to a safety limit of 95 per cent.

Excellent result (Group I) :

$$\delta = \sqrt{97 \frac{31}{97} \times \frac{66}{97}} = 4{\cdot}6, \text{ i.e. } 2\delta = 9{\cdot}2$$

Accordingly, an excellent result was found in 31 $\pm$ 9 cases corresponding to about 23–41 per cent.

Excellent result plus improvement (Groups I and II) :

$$\delta = \sqrt{97 \frac{70}{97} \times \frac{27}{97}} = 4{\cdot}4, \text{ i.e. } 2\delta = 8{\cdot}8$$

Thus, a beneficial effect of therapy was seen in 70 $\pm$ 9 cases corresponding to about 63–81 per cent.

In the following analysis of therapeutic results a distinction was made between in-patient and out-patients and an attempt was made to assess the influence of a number of factors on the results of therapy : sex and age, the type and extent of lesions, and the duration of disease.

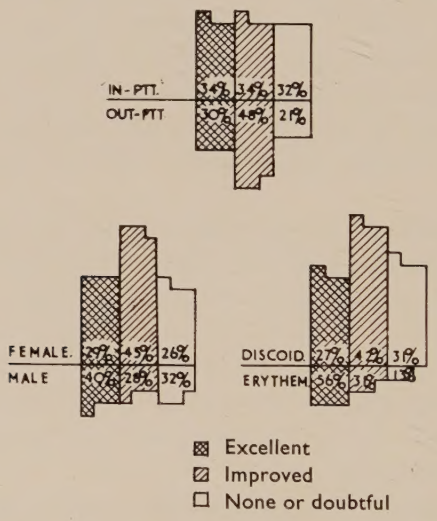

Fig. 2.

In-patients versus *out-patients* (Fig. 2).—If there was any difference at all, it was in favour of out-patients. The following analysis offers a likely explanation of this observation.

Sex.—There appeared to be no substantial difference in the results of treatment obtained in the two sexes. Men seemed to qualify for classification in the best group somewhat more frequently than the women, but the percentage of patients failing to respond to therapy was the same in both.

Type (Fig. 2).—The erythematous forms of the disease appeared to be somewhat more amenable to treatment than the discoid forms. This is in conformity with the general clinical impression. The number of erythematous cases (16) was too small, however, for tests of significance to be applied.

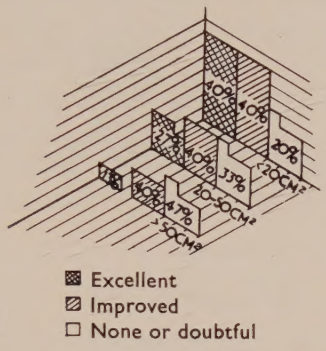

FIG. 3.

Extent of lesions.—The greater the area affected, the smaller the chance of a favourable result (Fig. 3). As this applied to both the erythematous and the discoid lesions, the two types will be discussed jointly. The difference was not significant, however ($\chi^2 = 3{\cdot}29$; $0{\cdot}10 > p > 0{\cdot}05$).

Duration.—The duration of the disease prior to mepacrine therapy seemed to have no substantial influence on the result (Fig. 4).

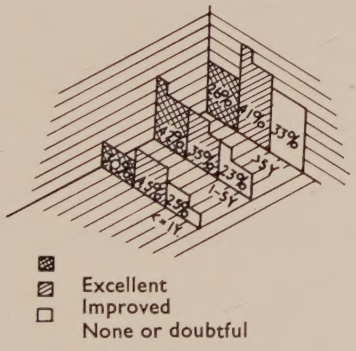

FIG. 4.

Age.—The patients were divided into three age-groups : under 45, between 45 and 60, and over 60. The reason for this division was that, in women, the first age-group corresponds to the age of sexual maturity, the second to the menopause and the immediate post-menopausal period, and the third to the senium. As conditions in the two sexes were very similar and the men were scantily represented, we did not divide the age-groups according to sex.

It was striking that the age-group over 60 showed by far the best results of therapy (Fig. 5). There were only 2 erythematous cases in his group. The difference must be regarded as significant ($\chi^2 = 3{\cdot}9$; $p < 0{\cdot}05$).

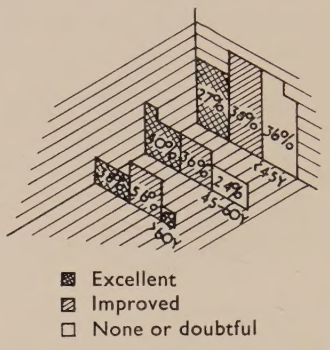

FIG. 5.

As mentioned previously (Fig. 2), results were somewhat better in the out-patients than in the in-patients. The classification of patients recorded in Fig. 1 showed a higher proportion of erythematous lesions in the group of out-patients than in the in-patients, and on the average the lesions were more extensive in the in-patients than in the out-patients. We think it likely that this explains why results were somewhat better in the out-patients.

Conclusions Regarding Therapeutic Results.

On the whole, our therapeutic results were in conformity with those reported by others. At a rough estimate an excellent result was obtained in one-third of the patients, improvement in another third, and a questionable or no result in the last third (Table II).

TABLE II.—*Lupus Erythematosus Treated with Different Remedies.*

	Remedy	Number of cases	A Excellent	B Improved	C None or doubtful effect.	Positive results per cent. (A + B).
Haxthausen, 1930	Auri chloride	37	12	17	8	78
Lomholt, 1935	Antileprole	31	11	12	8	74
Sterzi, 1950	Vitamin E	25	17	4	4	84
Black, 1953	PABA	32	3	8	21	34
Harvey and Cochrane, 1953	Bismuth	117	46	40	31	73
Harvey and Cochrane, 1953	Marphaside	56	20	17	19	66
Welsh, 1954	Panthothenic acid + vitamin E.	36	15	18	3	92

After a careful analysis of the results we concluded that the response was most favourable in the older age-groups, particularly the group over 60. Moreover, the prognosis seemed to be better in the erythematous than in the discoid lesions, though in the present material this difference was not significant. We found that the greater the extent of lesions the poorer the prognosis, but this difference also was not significant. Sex and duration of disease, on the other hand, seemed to have no influence on the result. Treatment was given at all times of the year, and there was a fairly even distribution of treatments all the year round. It was our impression that the result of therapy was somewhat poorer in the months of spring, but unfortunately we were unable to provide a numerical expression in support of this view.

Side-effects.

We aimed at producing yellow pigmentation of the skin, so that this was not regarded as a side-effect. We included only the side-effects which with some degree of certainty could be ascribed wholly or partly to mepacrine. The nature and incidence of side-effects are recorded in the following table :

	Women.	Men.	Total.	Suspension of therapy.	Incidence of side-effects (%).
Dyspepsia	12	4	16	7	17
Dermatitis	3	2	5	5	5
Disorders of sweating	7	1	8	0	8

Dyspepsia, which was the complication most frequently encountered, occurred in a total of 16 cases (about 17%). The incidence was the same in the two sexes, but the trouble was more severe in men. Thus, in two men the dyspepsia was so severe that medical treatment in hospital was required. In one man mepacrine treatment had to be stopped after two days. Dyspepsia often occurs after quite small or moderate doses ; in our cases treatment had to be stopped after the following total amounts : 0·4 g., 1·6 g., 7·4 g., 8·4 g., 9·8 g., 10·8 g., and 14·8 g.

Dermatitis occurred in 5 patients (about 6%) ; 2 cases were in the form of pityriasis (eczematoid), 2 were lichenoid, and one case had universal erythroderma. Treatment was stopped immediately in all these cases. The total amounts of mepacrine administered before treatment was stopped (onset of side-effects) were : 11·1 g., 11·7 g., 12·1 g., 13·7 g. and 19·9 g. The dermatitis cleared in all the patients after some months of treatment, and relapses have not been observed.

It should be mentioned that the one patient who had pityriasis simplex did not complete therapy for this complaint before mepacrine was started. We think it advisable to call attention to this fact so that all future patients are

examined for pityriasis simplex, which, if present, should be thoroughly treated before mepacrine is started.

Disorders of sweating were seen in 8 cases (about 8 per cent). Peculiarly enough, our patients complained mainly of an increased tendency to sweat. The fact is, apparently, that—unobserved by themselves—the patients developed a partial anhidrosis and noticed the compensatory excretion of sweat from intact areas of the skin. This disorder of sweating was followed by dermatitis in only one case. Our temperate climate and low summer temperatures seem to be responsible for the fact that this complication was rare in our patients and caused only slight inconvenience.

In none of our patients was mepacrine therapy seen to cause aplastic anæmia.

Relapses.

As mentioned earlier in this report, the period of observation after the end of therapy ranged between 3 months and 21 months ; the average was 9·7 months. The number of relapses seen during that period was fairly large. Table III shows the number of relapses and the time of their occurrence after discontinuation of therapy.

Table III.—*Time of Occurrence of Relapse in Relation to Time of Year.*

Month	Jan.	Feb.	Mar.	April.	May.	June.	July.	Aug.	Sept.	Oct.	Nov.	Dec
Number of patients	6	6	3	3	2	4	5	3	5	5	3	6

Time of Occurrence of Relapse in Relation to Discontinuation of Therapy.

Number of patients	5	10	12	4	3	2	5	4	3	4	0	1
Number of months after discontinuation of therapy	1	2	3	4	5	6	7	8	9	10	11	12

We saw no seasonal variation in the time of occurrence of relapse. Half the relapses occurred within about three months of stopping mepacrine, and none later than 12 months after discontinuation of therapy. Analysis according to therapeutic result shows that relapses occurred in 22 (about 71%) of the 31 patients in Group I (" excellent "), and in 29 (about 74%) of the 39 patients in Group II (" improved "). The average period to the occurrence of relapse was about 4 months in both groups.

As the period of observation was not the same in all the patients, it is likely that the relapse rate would be higher if the period of observation were extended to 12 months in all. The following is an attempt to estimate what the number of relapses would have been if the period of observation had been 12 months :

Period of observation	Number of patients.	Number of relapses.
Over 12 months	23	18 (78%)
6–12 months	34	27 (80%)
Under 6 months	13	6 (46%)

It will be seen from the above table that when the period of observation is about 12 months, the incidence of relapse is about 80%. It is therefore to be expected that relapses will occur in about 10 of the 13 patients in whom the period of observation was less than 6 months. Thus, the number of relapses to be expected in the whole series during 12 months will be 55 (80%).

It is not possible, on the basis of the present material, to predict whether the number of relapses will increase further if the period of observation is extended to 2 years. We saw no relapses later than 12 months after the end of therapy, but the number of patients observed for more than 12 months was only 5.

The Problem of Dosage.

As it is an exception to obtain complete freedom from symptoms during mepacrine therapy, the problem arises as to how long treatment should be continued. This question, which is not an easy one to answer, has a double aspect. First, for how long should treatment be continued if improvement does not occur : second, for how long should treatment be continued in cases showing improvement?

Kierland *et al.* (1953) state that if no result is obtained within 5 weeks, continuation of therapy is not worth while. It will appear from the table of doses administered (Table IV) that we continued treatment considerably longer than that (1 g. corresponds approximately to 1 week). It is, of course, extremely difficult to provide a numerical expression of the relationship between dose and therapeutic result. We made an attempt, however, to estimate the value of long-term therapy (high total dose) in the following way. A number of points are presented in the bottom row of Table IV. The number of points given for each group of doses was obtained by giving each case showing excellent improvement + 2 points, each case showing improvement + 1 point, and each case showing no improvement − 2 points. The sum of these points has been entered below the corresponding total dose. Positive figures were taken to indicate that continuation of therapy was worth while, and negative figures to indicate that continued therapy would be of questionable value. It will be seen that favourable results continued up to a total amount of 25 g. The possible value of still higher doses cannot be denied, but none of our patients showed improvement at doses higher than that. In other words, it is possible that the result would have been the same if treatment had been stopped at 25 g.

We conclude, first, that hope of improvement should not be given up until a total amount of at least 25 g. has been administered.

Second, that in cases showing early improvement treatment should not be stopped until a total amount of at least 25 g. has been administered. Even in the event of freedom of symptoms it is perhaps advisable to continue up to at least 25 g. in order to reduce the risk of relapse. These conclusions, of course,

assume that side-effects do not prevent continued administration ; if these are feared less energetic treatment may be preferable, but then the results of therapy are likely to be less satisfactory.

TABLE IV.—*Doses Administered.**

Dose (Grammes):	Under 5	5–10	10–15	15–20	20–25	25–30	30–50
Number of patients	3	22	21	24	8	8	11
Excellent improvement	0	10	7	10	2	2	0
Improvement	2	7	6	11	5	2	6
No or questionable improvement	1	5	8	3	1	4	5
Number of points†	0	+17	+4	+25	+7	−2	−4

* The approximate period of treatment will be obtained by calculating 1 g. = 1 week.

† The number of points was obtained by giving 2 points to each case showing excellent improvement, 1 point to each case showing improvement, and − 2 points to each case showing no definite sign of improvement.

LABORATORY TESTS.

To anticipate side-effects, numerous laboratory tests were performed as a routine measure.

Hæmoglobin (Hb) tests, determination of erythrocyte sedimentation rate (ESR), and thrombocyte and leucocyte counts were made on all patients before, during and after treatment. In addition, most patients were subjected to the following liver function tests : thymol turbidity test, Takata's test and determination of serum alkaline phosphatase, serum citric acid and serum proteins. The urine was examined for protein, sugar, urobilinogen, urobilin and bile pigment and studied microscopically. In most cases these tests were repeated several times during the course of treatment and at the end of treatment.

Some of the most significant results will be mentioned briefly :

Hæmoglobin.—Reduction of Hb levels (below 11 g. %) was seen only in a few patients, but no relationship with mepacrine treatment could be established.

Leucocyte and thrombocyte counts.—Individual tests disclosed leucopenia in 11 cases (below 4000/c.mm.), and 13 patients revealed definite evidence of thrombocytopenia (below 150,000/c.mm.). Three patients had both leucopenia and thrombocytopenia. No relationship could be established between these findings and mepacrine therapy.

No patient developed aplastic anæmia in response to mepacrine.

ESR.—Cochrane (1951) states that in the erythematous forms a low ESR is a factor determining a favourable prognosis. Conversely, if in the discoid forms the ESR is above 35 mm./hour, the prognosis is extremely poor.

As the present material contained very few cases of the erythematous type, the two forms of lesions were evaluated jointly and divided into three groups according to the ESR at the beginning of therapy.

TABLE V.

ESR at beginning of treatment (mm./hour.)	Number of patients.	Therapeutic result.		
		Excellent.	Improved.	Not improved.
0–10	40	12 (30%)	11 (28%)	17 (42%)
11–30	50	17 (34%)	25 (50%)	8 (16%)
Above 30	7	2 (28%)	3 (43%)	2 (28%)

It will be seen from Table V that no relationship was found between the ESR and the therapeutic result. In a number of patients the ESR rose temporarily during treatment, but here too a relationship between ESR and mepacrine treatment could not be definitely established. The distribution of ESRs was the same before and after treatment.

Liver function tests.—Thymol turbidity test was performed on 86 patients and found positive in 10 (above 0·15). Takata's test was performed on 85 and found positive in 7. 3 cases showed a positive reaction to both tests. Determination of serum alkaline phosphatase was made on 92 patients of whom 12 revealed values above 7 Buch units. Determination of serum citric acid was made on 65 patients of whom 53 had values above 27 μg. No changes were observed during treatment. Determination of serum proteins was made on 95 patients. The most frequent observation was a change in globulin content. 22 revealed values below 2·0 per cent, 4 above 3·5 per cent.

The outcome of these comprehensive laboratory examinations was incommensurate with the effort. Thus, the changes elicited by these examinations could not be demonstrated to bear any relation to mepacrine treatment. Changes in laboratory findings directly ascribable to mepacrine were seen in only one patient in whom, concurrently with the onset of dermatitis, Takata's test became positive (+ — +) and the thymol test rose from normal values to 0·43.

The other positive findings seemed to reflect only the lesions of the original disease.

TUBERCULIN SENSITIVITY.

Tuberculin reaction tests were performed on a great number of the patients to determine the tuberculin sensitivity in patients with lupus erythematosus. The method employed was the graduated Mantoux test with old tuberculin advocated by Bonnevie (1934). The results reported by Bonnevie and Björnstad (1940) are listed for comparison.

L.E. Patients without Recognized Tuberculous Disease.

	Authors' study.	Bonnevie & Björnstad (1940).
Mantoux negative	7	2
Mantoux positive	77	66
Not tested	13	—

Tuberculin Positive.

Dose.	pTb.	Authors' study.	Bonnevie & Björnstad (1940) L.E. patients.	Bonnevie & Björnstad (1940) Controls.
1 mg.	0	8 (10%)	5 (7·5%)	39 = 5%
0·1 mg.	1	20 (26%)	19 (29%)	211 = 25%
0·01 mg.	2	28 (36%)	21 (35%)	260 = 31%
0·001 mg.	3	14 (18%)	10 (15%)	178 = 21·5%
0·0001 mg.	4	7 (9%)	5 (7·5%)	78 = 9·5%
0·00001 mg.	5	0 (0%)	6 (9%)	69 = 8%

Our results agree with those of Bonnevie and Björnstad that there is no evidence that lupus erythematosus influences the sensitivity to tuberculin.

Discussion.

The problem of effective treatment of lupus erythematosus remains unsolved. Mepacrine seems to be fully comparable with other drugs, such as gold and bismuth, but on the other hand it does not offer much more than these. The number of side-effects is not inconsiderable, and especially the risk of severe dermatitis must be taken into account. In addition, there is the cosmetically unpleasant but otherwise harmless yellow pigmentation of the skin.

It was striking that the prognosis was most favourable in the older age-groups, particularly in the group over 60 years. Although no explanation can be offered of this it seems worth mentioning as it may give guidance in future studies.

The clinical investigations here reported did not contribute much to elucidate the method of action of mepacrine.

In the patients included in the present study the incidence of relapse was 80% during an observation period of 12 months.

The high incidence of relapse seems to indicate that the effect of mepacrine is symptomatic and that the permanent cure obtained in a few cases is ascribable only to a supportive effect on the reparative processes already taking place.

In the future, attention must be concentrated mainly on chloroquine, with which excellent results have already been reported by several investigators (Kierland *et al.*, 1953 ; Harvey and Cochrane, 1954 ; Pillsbury and Jacobson, 1954 ; Rogers and Finn, 1954). The advantages of chloroquine over mepacrine are that it does not produce yellow pigmentation of the skin, and that the side-effects seem to be fewer. It will be interesting to see whether long-term therapy of lupus erythematosus with chloroquine in small doses will be capable of preventing the tendency to relapse.

Summary.

In 1953 and 1954, 97 cases of lupus erythematosus (81 discoid and 16 erythematous) were treated with mepacrine. The total dose ranged between 1·6 and 49·8 g., and the average dose was 18 g.

Of the 81 discoid cases treated, 22 (27%) showed an excellent result, 34 (42%) improved, and 25 (31%) were unchanged or doubtful. Of the 16 erythematous cases treated 9 (56%) obtained an excellent result, 5 (31%) improved and 2 (13%) were unchanged or doubtful.

A little more than half of the patients were treated in hospital. These patients showed no better result than out-patients.

Sex and duration of the disease seem to have no influence on the result of therapy. Large extent of lesions seems to indicate an unfavourable result, but the difference is not significant.

The prognosis was most favourable in the older age-groups. Over 60 years of age an excellent result was obtained in 6 (38%), improvement in 9 (56%) and only 1 (6 %) failed to improve.

The number of side-effects was not inconsiderable. Dermatitis occurred in 5 cases, dyspepsia in 16 and disorders of sweating in 8.

After stopping treatment most of the patients were observed for about 12 months. The number of relapses during the period of observation was about 80%. The frequent relapses seem to indicate that the effect of mepacrine is symptomatic rather than curative.

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AN OUTBREAK OF LINEAR SCARRING ALOPECIA.

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AND

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In 1954 a peculiar type of scarring alopecia of the scalp appeared in all 7 members of a Johannesburg family. This resembled the alopecia parvimaculata of Dreuw (1910*a*, *b*) but differed from this and similar epidemics in several important features. We were able to perform more elaborate investigations than have been reported previously and have therefore described this outbreak in some detail.

The chief difficulty in comparing our cases with those of previous epidemics lies in the basic distinction between the cicatricial and non-cicatricial alopecias (Sabouraud, 1929*a*). It will be seen that these epidemics are predominantly of the temporary, non-cicatricial type, while all our cases resemble pseudo-pelade in many respects, being essentially cicatricial.

Reports of contagious alopecia date from the first half of the last century and have caused much confusion. Even after ringworm had been recognized and placed in a separate category, reports of epidemics of alopecia in institutions and in the forces were frequent, especially in France. Both Sabouraud (1929*b*) and Bowen (1915) mentioned these epidemics as being recorded since the days of Hardy, Besnier and Foulard, but Sabouraud at least was sceptical. Sabouraud (1929*c*) also mentioned the distinction between contagious and trophoneurotic patchy baldness: the former he attributed principally to scarring after impetigo. As far as true alopecia areata is concerned, he stated categorically that it does not exist in epidemic form ("*Les épidémies de pelades n'existent pas*").

Pseudo-pelade is the term given to a peculiar, patchy, cicatricial alopecia without gross signs of inflammation (Photinos, 1930). We have seen no reports of pseudo-pelade being transmitted as a contagious disease. Further, we have been unable to find any mention of a contagious, *scarring* alopecia, other than those caused by fungal infections such as favus or (according to Sabouraud) impetigo, except as claimed by Dreuw (1913) in his last publication.

Alopecia parvimaculata.

The following accounts refer to outbreaks of patchy alopecia which, except in (*e*) below, were certainly not caused by infection with a fungus.

(*a*) In 1891 Bowen (1915) observed the first of two epidemics of alopecia in an institute in Boston. Sixty-three of 69 children developed numerous round bald areas on the scalp, smaller than those of alopecia areata. Some areas showed jagged, angular and irregular outlines. Histologically, there was " atrophy of the pilo-sebaceous follicles ".

Six years later a second, similar epidemic occurred in the same institute : Bowen thought that this was the result of the return, as an adult, of one of the children affected in the first outbreak. In no case was atrophy noted and permanent baldness did not result, as far as Bowen could ascertain. Regrowth usually took place in 6 months. Bowen at first assumed that he was dealing with epidemic alopecia areata, but later believed that his cases were identical with those of Dreuw (*infra*).

(*b*) Dreuw (1910*a*, *b*) described two outbreaks of epidemic alopecia in a Berlin orphanage, each affecting about two-thirds of the inmates. Most lesions occurred on the sides and back of the head ; circular lesions were rare, the majority being irregular, spindle-shaped or angulated. Stumps of hair were present in the affected parts. As complete recovery took place in 90% of cases Dreuw assumed that local antiseptic treatment had been curative. He proposed the name alopecia parvimaculata for the condition he described.

In a later publication Dreuw (1913) stated that alopecia parvimaculata was the first stage of alopecia atrophicans (pseudo-pelade). He recognized an " atrophicans " and " non-atrophicans " variety of alopecia parvimaculata : the latter presumably comprised the 90% of cases which recovered completely. He did not mention whether in any particular case only some of the lesions recovered while others progressed to alopecia atrophicans : his series of photographs showed the transformation of a few, discrete areas into the well-known severe and confluent lesion of pseudo-pelade. He also quoted an epidemic described by Boegershausen (1912) in Lüdinghausen : this consisted of 101 cases, all of whom were " cured "—presumably with regrowth of hair. This epidemic was regarded by Dreuw as an example of alopecia parvimaculata non-atrophicans.

It is most unfortunate that no histopathological investigations were reported from any of the cases seen by Dreuw, or referred to in his publications, for only in this way could it have been proved that his " atrophicans " and " non-atrophicans " types were examples of the same disease.

(*c*) Colcott Fox (1913) saw an epidemic of 21 cases in an institution in England. Most patients presented only one patch of baldness, but one had three. The lesions were usually round or oval, but 2 were lanceolate and 1½ inches long. The

edges were not circumscribed: many of the patches showed follicles plugged with dark, pigmented debris. In passing Colcott Fox mentioned a similar epidemic observed by Hillier (1865) at the Hanwell Schools.

(*d*) Davis (1914) saw two epidemics in an orphanage, affecting 216 persons, of whom 3 were adults. In his cases the patches were covered by short stumps. He noted also an opaque swelling around the root sheath.

(*e*) Werther (1931) described an epidemic of alopecia parvimaculata in Dresden associated with an epidemic of Microsporum ringworm. The manifestations of both conditions appeared and disappeared at about the same time. About 400 children were affected. Most of the lesions were round or irregular, but a few were linear. The affected skin was smooth, white and shiny and there was usually no trace of follicles visible. All the children had occipital adenopathy. Werther thought that the lesions of alopecia parvimaculata, occurring in association with ringworm of the scalp, represented a microsporid.

Case Reports.

The family here described consists of 7 people, Mr. and Mrs. M— and their 5 children. The parents are both of Greek extraction, the father and children having been born and brought up in South Africa. An eighth subject, Mrs. M —'s brother, who stayed with the family for some months, is said to be similarly affected, but has not been seen by us. The family are in comfortable circumstances, the home is clean and the children well cared for. The parents are exceptionally intelligent and have been able to give clear accounts of the history and appearances during the early stages of the outbreak.

The first lesion was noticed in March 1954, and by October every member of the family was affected. Table I summarizes the progress of the outbreak. During the seven months that have elapsed since Janaury 1955 fresh lesions have continued to appear, at the rate of one or two every month in all the patients.

Table I.—*Development and Progress of the Outbreak.*

Name.	Age.	First lesion seen (1954).	Number of lesions in January 1955.
Christopher	6	March	11
Paul	13	May	10
Gregory	10	May	2
Alexander	8	May	3
Mrs. M—	34	July	3
Mr. M—	47	August	4
Helen	5	October	1

Subjective symptoms.—The adults and older children state that a mild itch is noticed for 3 or 4 days, and that loss of hair occurs about a week later.

Objective signs.—Mrs. M— pointed out that the first noticeable change was that the hair at the site of a fresh lesion "stared", i.e. would not lie flat. She states further that a faint pink flush was visible in some cases, but we have been unable to confirm this. Within a few days the hairs had fallen, leaving a smooth bald area at first normal in every respect except for the absence of hair; after 3 weeks the area was atrophic, smooth, white and shining. Follicular openings could be distinguished with difficulty and in another month were invisible. The patches were not depressed more than is

normally seen following epilation : Sabouraud has pointed out that the actual bulk of hair substance removed by epilation is sufficient to cause this slight depression.

Of the 34 lesions seen in January 1955 only three were roughly circular (Fig. 1) ; the rest were linear, measuring from 1 to $1\frac{1}{2}$ inches in length and about $\frac{1}{8}$ inch in width (Fig. 2). All parts of the scalp were affected : on the sides and back the linear lesions invariably pointed to the vertex. The general impression was that of a healed cut, possibly best described in French as *alopécie en petits coups de sabre*. At no time were broken stumps or plugged follicles seen, as were reported in the epidemics of Dreuw, Colcott Fox and Davis.

Fig. 1.—Mrs. M—. A rare, non-linear lesion.

During the period of hair-fall macroscopic examination of the hairs showed these to be either broken above the root, or to be surrounded by a white gelatinous sheath for about $\frac{1}{8}$ inch at the proximal end. Microscopic examination of these latter hairs showed the presence of a fairly clear cut break at about the middle of the root, the shaft, upper portion of the root and its sheath showing no abnormality. None of these patients showed any other abnormality or disease of the integument.

In June 1955, Mrs. M— first noticed diffuse thinning of Paul's and Christopher's hair. Examining Christopher in August we could not convince ourselves of this, but are inclined to believe Mrs. M – since she has shown that she is a reliable observer.

Treatment.—The four male children were given a daily shampoo with a soap containing 3% of hexachlorophene. Fresh lesions continued to appear both during and after a month's trial of this method. Three of these cases were subsequently treated with inunctions of an ointment containing $\frac{1}{2}$% of neomycin sulphate twice daily to all affected areas in all stages. This treatment was suggested by the results of bacterial culture and sensitivity tests detailed below. The progression of existing early lesions and the appearance of fresh areas were not prevented.

A sporadic case.

Recently a man of 21 was seen by one of us for urticaria which had been present for 5 years. During routine examination 3 lesions of linear, scarring alopecia were

found, resembling in every way those presented by the M— family. In addition it was thought that the hair of the upper occipital region was diffusely thinned. The patient said that he had first noticed the linear alopecia about six months before and that no other member of his family was affected. It was not possible for us to examine these, as the patient had come from a distance of over 100 miles.

Fig. 2.—Christopher M—. The common, linear lesion, pointing to the vertex.

Laboratory Investigations.

Mycological.—Hairs were taken from recently affected areas for examination. These were all loose and many showed a gelatinous sheath. Direct microscopic examination in 20% potassium hydroxide was negative on 2 occasions in hairs taken from Mr. and Mrs. M—, and once in hairs taken from Christopher, Gregory and Paul.

Hairs from these 5 patients were planted on dextrose agar, Littman's oxgall agar and Actidione medium. No growth was observed after 4 weeks' incubation at 23° C.

Bacteriological.—Hairs from Paul were taken for bacterial culture and a non-haemolytic *Staphylococcus aureus* was grown. More recently cultures made from Christopher's hair resulted in the growth of a haemolytic *Staphylococcus aureus*, a non-haemolytic *Staphylococcus aureus* and a *Staphylococcus citreus*. *In vitro* sensitivity tests showed that all three organisms were sensitive to penicillin, aureomycin, terramycin, erythromycin, achromycin, tetracyn and polycyclin in all dilutions, and bacitracin and neomycin were judged suitable for topical application.

Histopathology.—Under procaine anaesthesia a 4 mm. punch biopsy specimen was taken from Mrs. M—'s scalp. At the time there was no visible lesion, but she stated that there had been mild itching a few days before and unerringly marked the biopsy site with a spot of ink.

The specimen was fixed in 4% formalin and embedded in paraffin wax. 240 serial sections were cut at 6μ and stained with haematoxylin and eosin.

Examination of these sections shows a normal epidermis, with no atrophy, follicular plugging or degeneration of the basal cell layer. The dermal collagen stains normally,

individual collagen bundles are of normal thickness and density and they are not increased in number. The sweat glands show no abnormality. Arrector pili muscles are well developed and are normal in appearance. The blood vessels are somewhat dilated but their walls show no pathological changes.

The major pathological process in these sections is confined to a single follicle and appears to have originated in its middle third. The lower third of the follicle is atrophic, shrunken and grossly distorted. The papilla is not recognizable. Embedded in the

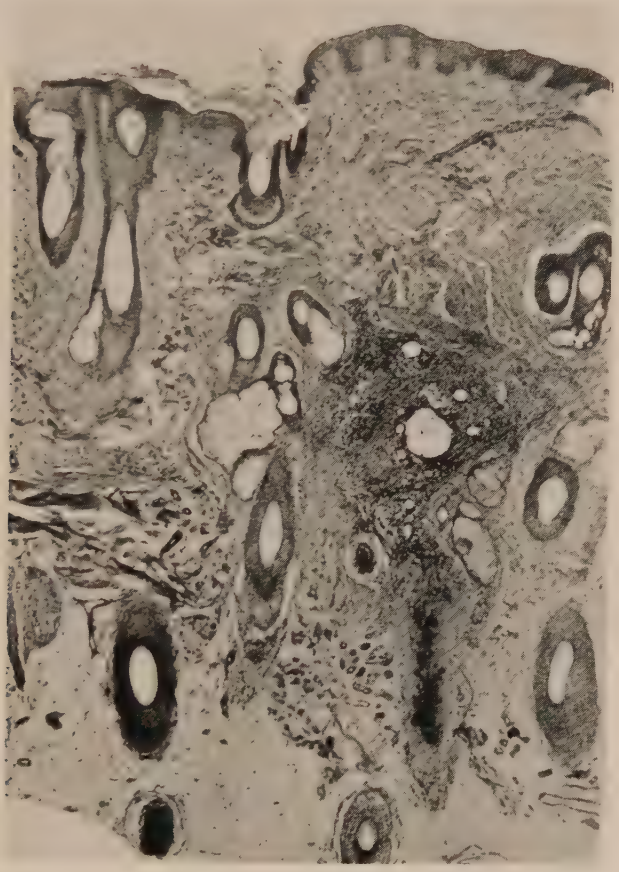

Fig. 3. –H. & E. ⁄ 45.

remnants of the follicle there are scattered granules of melanin pigment but no evidence of a hair root. There is no cellular infiltrate around it.

In the middle third at about the level of the sebaceous glands the hair follicle disappears into a granulomatous lesion (Fig. 3), in the centre of which is a large cyst-like space containing some amorphous basophilic material (Fig. 4). Similar smaller cyst-like spaces are scattered throughout the granuloma. The large space and several of the smaller ones are surrounded by endothelial cells and histiocytes, some of which are large and multinucleated, and many have a foamy cytoplasm. The general appearance is reminiscent of fat necrosis. A few multinucleated giant cells with peri-

pheral nuclei are present. The outer zone of the granuloma consists of lymphocytes. No polymorphonuclear leucocytes or plasma cells are seen.

The shape of the lesion is roughly spherical with large, irregular protuberances. These protuberances have a similar histology to the central mass but when cut in cross-section their appearance is typically tuberculoid.

From the periphery of the granuloma the lymphocytic infiltrate extends for a considerable distance along the blood vessels which enter or leave it.

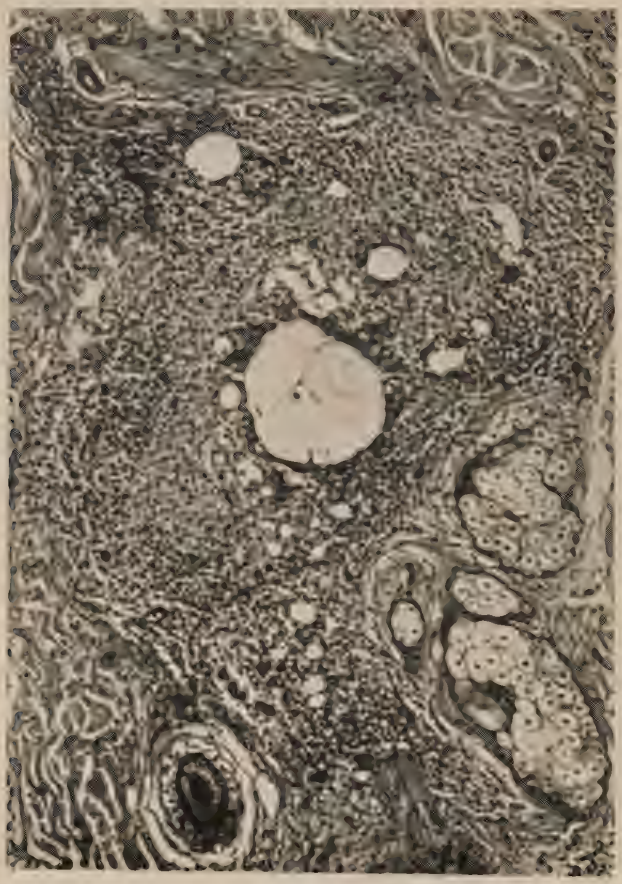

FIG. 4.—H. & E. × 120.

No fragments of keratin or of disintegrated hair shaft could be seen. The upper third of the affected follicle above the granuloma is empty and is free of inflammatory infiltrate.

Most of the neighbouring follicles show no abnormality but a few are surrounded by a scanty lymphocytic infiltrate.

Neither fungal elements nor bacteria were observed in sections stained with McManus, Gram or Ziehl-Nielsen stains.

The histological features are those of disintegration of the middle third of the follicle and sebaceous glands with loss of the hair. Liberation of sebaceous material

has presumably stimulated the formation of a tuberculoid granuloma. The lower third of the follicle has atrophied. There is nothing in the histological picture to suggest the cause of this disintegration, but the absence of polymophonuclear leucocytes and plasma cells suggests that pyogenic organisms do not play a part.

Later, a second 4 mm. punch biopsy specimen was taken from another lesion on Mrs. M—'s scalp; this lesion was several months old and atrophic. The excision was

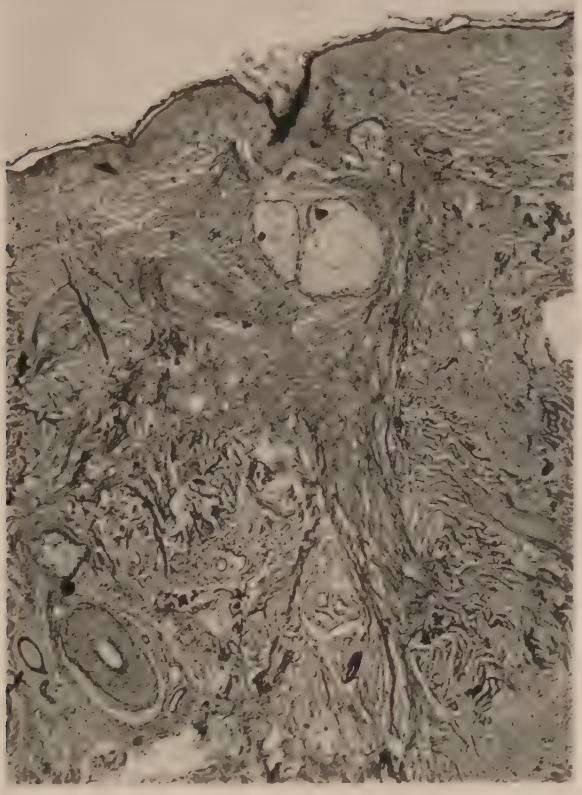

FIG. 5.—H. & E. × 45.

planned to include the edge of the lesion. This specimen was treated in the same way as the first one.

Examination of the sections shows no change in the epidermis. In the dermis there is a band of dense collagen bundles running at right angles to the surface, contrasting with the normal reticular, horizontal pattern (Fig. 5). Normal elastic tissue distribution stops abruptly at the margin of this area, which obviously represents a cylindrical, perpendicular scar.

DISCUSSION.

The occurrence, within a few months, of alopecia in every member of a family convinces us that we are dealing with a contagious condition. The parents are

not blood relations and an hereditary condition would, in any case, hardly attack persons of widely different ages within a period of a few months.

Our cases do not correspond with any of those previously described, with the possible exception of some of the cases mentioned by Dreuw. It is true that the epidemics detailed above have a superficial resemblance to ours, the chief difference being their benign progress with almost invariable regrowth of hair. Such regrowth we believe to be impossible in our cases, basing this belief on our clinical observations and on the histological evidence of scarring after destruction of the pilo-sebaceous apparatus. We therefore classify our cases among the cicatricial alopecias and, in view of their histopathology, consider them to be a form of pseudo-pelade. The main features of this and previous outbreaks are summarized in Table II.

TABLE II.—*Comparison of Principal Features in 6 Reported Outbreaks of Contagious Alopecia.*

	Bowen 1891–1897	Dreuw 1910–1913	Colcott Fox 1913	Davis 1914	Werther 1931	Loewenthal and Lurie, 1955.
Number of cases . .	89	Over 200	21	216	400 (approx)	7
Round areas	Majority	Rare	Majority	—	Majority	Few
Irregular areas	Yes	Yes	Yes	—	Yes	No
Linear lesions	Few	A few spindle shaped	2 lanceolate	Presumably none	Few	Majority
Plugging of follicles	Not mentioned	Short stumps	Yes, with debris.	Short stumps	No	No
Histology .	Atrophy of piloseba-ceous follicles	—	—	—	—	Pseudo-pelade
Permanent baldness	Regrowth in 6 months	In 10%	Not mentioned	Not mentioned	Rare	Yes

Civatte (1930) describes, under pseudo-pelade, the following histological appearances : invasion of the follicular epithelium by the infiltrate, shedding of the hair with a white sheath, the presence of lymphocytes, epithelioid and foreign-body giant cells, the disappearance of the sebaceous glands without a trace, and the replacement of the pilo-sebaceous apparatus by a cylindrical scar, clearly shown with an elastic tissue stain. It will be noted that our histological material shows all these features, except the first ; this discrepancy is presumably due to our biopsy material being taken at a somewhat advanced stage.

Sabouraud (1929), Photinos (1930), Desaux (1953) and Laymon (1947) all classify alopecia parvimaculata under pseudo-pelade, possibly because Dreuw (1913) himself, as stated earlier, eventually believed that his alopecia parvimaculata represented an early stage of alopecia atrophicans (pseudo-pelade). It is difficult to reconcile this view with his earlier report of a recovery rate of 90%. If, however, it is correct, then we shall be compelled to differentiate a

contagious type of pseudo-pelade, with lesions following each other in rapid succession, from the usual non-contagious pseudo-pelade with its slow evolution. In our cases, the linear form of the great majority of lesions seems to distinguish them from those of previous epidemics, in which only an occasional linear or lanceolate lesion was seen.

A further difficulty arises from Dreuw's concept (without a histopathological basis) of " atrophicans " and " non-atrophicans " varieties of alopecia parvimaculata. If our cases are identical with Dreuw's " atrophicans " variety (in spite of the clinical differences shown in Table II), then his observation of the eventual development of pseudo-pelade fits our histological findings. But how, in that case, can we account for his 90% of cases of alopecia parvimaculata non-atrophicans ? We find it difficult to believe that hair-loss consequent on this type of pathological alteration could ever be reversible, and in none of our cases has there been evidence or, we would like to add on clinical grounds, likelihood of regrowth of hair.

We suggest, therefore, that Dreuw confused two separate entities ; First, an alopecia parvimaculata non-atrophicans identical with the outbreaks described by Bowen, Colcott Fox, Davis, Boegershausen and possibly Werther : these accounted for 90% of his cases. And second, an alopecia parvimaculata atrophicans, comprising 10% of his cases and representing an early stage of pseudo-pelade. This second group is the only entity with which we feel that our cases could be equated, if we ignore certain differences in the clinical picture.

It would perhaps be best to adopt from Degos, Rabut, Duperrat and Leclercq (1954) the concept of a Pseudo-peladic State. Having noted that lesions clinically and histologically typical of pseudo-pelade could accompany or succeed such diseases as lupus erythematosus, lichen planus and scleroderma, these authors contend that pseudo-pelade should no longer be regarded as a primary specific disease entity : they prefer to regard it as a follicular or perifollicular process terminating in scarring, secondary to a number of varied dermatoses. If we include alopecia parvimaculata among these, then most of the above nosological difficulties would be resolved.

Summary.

An outbreak of contagious, cicatricial alopecia is described. The great majority of lesions were of linear shape. All seven members of a family were affected. Another sporadic case has also been seen.

The histological appearances are described in detail ; no such description is available from previous outbreaks.

It is suggested that this outbreak may be identical with a minority of the cases described by Dreuw (1913) as alopecia parvimaculata and representing an early stage of the pseudo-peladic state.

We gratefully acknowledge the assistance of Drs. J. Gluckman and W. Lewin, of Johannesburg, who prepared some of the histological material and performed some of the mycological and all the

bacteriological work. Mr. Ulrich of the S.A. Inst. for Medical Research prepared the microphotographs.

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MATTING OF THE HAIR BY SHAMPOO

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THE following case is sufficiently uncommon to deserve notice.

A housewife aged 51 washed her hair with a proprietary cream shampoo which she had never used previously. Within a few minutes of using the shampoo her hair became tangled so that it was impossible to unravel it.

She was in good health and had not had any previous disease of the hair or scalp. She had never used bleach, dyes or setting lotions, nor had any permanent wave applications. Her hair had never been cut. It was particularly long and dense, grey-brown in colour, reaching to her waist, and of fine texture.

When she was seen three weeks after the application of the shampoo there was gross matting of the hair, resembling felt, at the occiput. In adjacent areas the hair was less tangled and on the rest of the scalp it was normal. It was necessary to cut off a large mass of the hair since it was impossible to disentangle it. Subsequent regrowth was normal, although the patient states that her hair is straighter than it was.

Macroscopic and microscopic examination of the affected hairs showed no abnormality. The unaffected hair was long and wavy, with twisting of the hair shaft to 180° at intervals of 6–9 inches. This twisting was more frequent, at about 2–3 inch intervals, in the matted hair.

The chief ingredients of this cream shampoo are wool fat, sodium primary alkyl sulphates, chiefly lauryl sulphate, sodium alginate, sodium chloride and perfume. About 500,000 of these shampoos are sold annually, and it is possible that several millions of shampoos of similar type are used each year. None of the manufacturers in Great Britain has had reports of a similar occurrence.

Graham (1953) reported a case in the United States of America of a girl aged nine years, in whom matting of the hair at the occiput occurred while using a liquid lauryl sulphate shampoo. In his patient it was impossible to reproduce the matting effect with hair from other regions of the scalp. The photographs from his report and from the case described here show a similar structure of the hair, matted together in tufts.

It seems probable that the occipital hair was affected in each case because it is common practice to apply the shampoo directly to this area before rubbing it over the whole scalp, as the head is held over a basin.

I wish to thank Dr. H. J. Wallace for permission to publish this case and for his encouragement. The photographs were taken in the Department of Photography, St. Thomas's Hospital Medical School.

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ROYAL SOCIETY OF MEDICINE

SECTION OF DERMATOLOGY.

President—Dr. Reginald T. Brain.

21 April 1955.

Kaposi's Haemorrhagic Sarcoma.—Dr. R. H. Marten (for Dr. J. E. M. Wigley and Dr. H. Haber).

A West African, aged 34 years. Occupation : Scientist.

Case history.—In 1950 while living in Liberia his feet began to swell. This was attributed to filariasis and he was treated with atebrin injections with apparent improvement. In April 1954 an itching eruption developed on the feet which became more swollen and at times painful especially on rising in the early morning. During the next two months a rash appeared on the penis and scrotum. In December 1954 painful swelling of the backs of the hands and fingers developed.

Past history.—Yaws as a child. Jaundice 1945.

Family history.—Nil relevant.

Previous treatment : 1950 : Six injections of atebrin.

April 1954 : Penicillin injections for two weeks followed by 6 injections of bismuth in Paris.

December 1954 : Banocide 200 mg. daily for two weeks for treatment of filariasis.

Examination.—There is brawny thickening and pitting oedema of the lower part of both legs and feet, and some swelling of the backs of the hands. Adherent warty thickening is present on the toes of both feet—more on the left foot.

Over the dorsa of the feet are some firm nodules and pigmented plaques. A pigmented area is also present on the ulnar border of the hand. There are similar lesions on the right thigh and sides of the scrotum. On the sole and medial aspect of the left foot are smooth round purplish nodules of varying size and elevation. Scarring is present on the front of both legs. No abnormality was detected in any other system.

Investigations : (1) Hb 88%. RBC. 5·3 M. P.C.V. 42%. Slight hypochromia.

(2) WBC. 6500. Neutrophils 58% ; lymphocytes 36% ; monocytes 5% ; eosinophils 1%.

(3) Platelets : 210,000 per cmm.

(4) ESR. : 19 mm./hr.

(5) Blood Wassermann and Kahn tests : negative.

(6) Skiagrams of legs and feet : Calcification in the feet suggestive of loa-loa ; otherwise no abnormality.

(7) X-ray chest : N.A.D.

(8) Histology (from right foot) shows a cellular lesion in the dermis composed of spindle cells with vascular channels, haemosiderin and a few foam cells.

Treatment.—He has received 27 million units of crystalline penicillin in a dosage of one to two million units daily intramuscularly. Initially, while in hospital, he noticed much less pain in the legs but this has now returned and new nodules have appeared in spite of continued treatment.

No radiotherapy has been used in this case.

Discussion.—In the past 10–12 years a number of cases of Kaposi's sarcoma have been reported in Africans (Persky and Lisa, 1944; Dennison and Evans, 1946; Kaminer and Murray, 1950). Kaminer and Murray (1950) reviewed the files of the South African Insitute of Medical Research for the period 1942–49 and found 38 histologically proved cases.

References.

Persky, B. P. and Lisa, J. R. (1944) *Arch. Derm. Syph., Chicago*, **49**, 270.
Dennison, W. and Evans, W. (1946) *Tr. Roy. Soc. Trop. Med. Hyg.*, **39**, 521.
Kaminer, B. and Murray, J. F. (1950) *South African J. Clin. Sc.*, **1**, 1.

Dr. J. C. Belisario: This case interested me particularly because of the large doses of penicillin given without effect, especially as I have in mind several reputable workers who claimed to have obtained beneficial results from its use. I would be interested to know if there was any beneficial result from atebrin administration. I should be similarly interested in the use of chloroquine which has been claimed to have been useful in this condition.

Dr. L. Forman: Some cases of Kaposi's disease of the legs are preceded by or associated with considerable solid oedema of the affected limb. This recalls the somewhat analogous phenomenon of angiosarcoma of the limb (Stewart-Treves syndrome) after removal of glands for a carcinoma of the breast. The patient shown today gives a history, real or suspected, of filarial infection. Perhaps in some cases of Kaposi's disease the lymphatic obstruction may occur primarily, and if caused by micro-organisms the response of Kaposi's disease to penicillin may be thus explained.

Dr. H. Haber: Whereas in the Stewart-Treves syndrome lymphangiosarcoma follows only surgical lymphoedema due to mastectomy, in Kaposi's sarcoma oedema and tumour formation occur side by side.

Generalized Morphoea.—Dr. Stephen Gold.

Mr. A. B., aged 70, has suffered for the past six or seven winters from bronchitis with breathlessness and for some two years there has been a tremor of the hands. Eighteen months ago he noticed swelling of the ankles associated with redness and irritation of the skin which became scaly; soon afterwards the skin became tight and shiny, and this process spread upwards over his legs and thighs and also to his trunk starting at the lower abdomen and reaching the chest. More recently the upper arms have been patchily involved. As a result there has been some limitation of movement of the legs and chest. The hands, forearms and face are free; there is no Raynaud's phenomenon and he does not suffer from dysphagia.

He was first admitted to hospital over a year ago when his condition was:

Marked breathlessness, wheezing and orthopnoea. The skin was red, thickened and shiny over feet, legs, shins, thighs, abdomen, back and, to a lesser extent, his chest and upper arms. The muscles of his legs appeared wasted and there was some pitting oedema and tightness of the skin, particularly of the legs. His face was normal and there was no telangiectasia. The blood pressure was 190/100. No clubbing of the fingers, but the chest expansion was limited and the signs in his chest were consistent with chronic bronchitis and emphysema. The abdomen was tense and resonant, but there was no ascites and no mass or viscus was palpable. There was a left hydrocele.

Past medical history shows that he has been subject to recurrent urticaria since the first World War. He had amoebic dysentry in 1918 and was admitted to hospital in 1942 for investigation of dyspepsia. In the last few years he has suffered from a left hydrocele.

Investigations at this time revealed a normal blood picture. Sedimentation rate: normal. Sputum showed *H. influenzae*. Serum proteins: a slight increase of the globulin fraction. Liver function tests were within normal limits. Blood urea: 24 mg.%. Barium swallow: normal. Chest skiagram showed some left ventricular hypertrophy. Skin histology consistent with scleroderma. E.C.G. showed flattened T waves.

During his first admission to hospital on account of his chest condition he was treated with penicillin, but after this the skin erythema and irritation increased with development of urticaria. On stopping penicillin these symptoms improved, but the skin thickening remained the same. A course of systemic cortisone was started; he received 200 mgm. daily for two weeks and was discharged receiving 100 mgm. daily for a further two weeks. This produced no appreciable alteration.

Recent investigations have again shown no abnormality with routine liver function tests and the barium swallow taken in the prone position showed no abnormality. Chest skiagram showed slight ventricular enlargment and prominence of the aortic knuckle. Examination of sputum was non-contributory other than a strain of *Haemophilus influenzae* which was sensitive to the usual antibiotics. Complete blood count: Haemoglobin, 14·4 g. (97%); white blood cells, 10,000 per cu. mm.; polymorphs, 75%; eosinophils, 1%; lymphocytes, 18%; monocytes, 6%.

Serum proteins; albumin 2·8, globulin 4·1 (A/G ratio 0·7). Electrocardiogram shows low voltage curves and there were abnormal T wave patterns.

Biopsy sections from skin and muscle of the leg show epidermal flattening with loss of papillae; the most superficial layers of the dermis have an unduly homogenous collagen with some lymphocytic infiltration mainly but not entirely perivascular and periglandular. There is no strangling of glands and no significant vascular change.

The muscle does not show any evidence of fibrosis or atrophy, such as may be found in scleroderma.

Progress.—He has been treated in the physiotherapy department so as to increase mobility and encourage a softening of the skin—so far with little change. The local application of hydrocortisone ointment to a limited area of skin for three weeks produced no apparent change in the underlying skin texture.

Dr. G. B. Dowling: I agree with Dr. Gold's classification of this case. The term generalized scleroderma may be applied equally to progressive symmetrical scleroderma with its varied systemic manifestations, or to this case in which as far as we can judge from the facts as far as they are known at present, no systemic developments are to be expected. The lesions are those of morphoea but they are distributed widely all over the body. I prefer to use the term generalized morphoea for this condition and scleroderma or progressive scleroderma for the other.

The President: I prefer to regard morphoea as a localized condition. In contrast was a South African with a compound fracture of the leg who developed scleroderma around the site of the fracture; it finally involved the whole body and the patient died of pneumonia. The localized type sometimes resolves and therefore I hope some members will tell us how we may avoid the confusion of terms.

Dr. I. B. Sneddon: I would support the diagnosis of generalized morphoea as it is very like a case shown to this Society by Dr. R. Church which involved the tongue and the skin but had no visceral lesion.

Reference.

Church, R. E. (1953) *Brit. J. Derm.*, **65**, 415.

Dr. Dowling: Prognosis varies considerably, recovery taking place in the course of three or four years in a proportion of them, leaving what looks like atrophy rather than sclerosis. Cases of more limited distribution on the other hand, particularly the band form which is so often associated with underlying muscular atrophy, seem sometimes to persist indefinitely, though of course some localized cases also get well.

Dr. P. J. Feeny: I think this case should be called morphoea. It is better to make a distinction between those cases that are systemic and those that are not, no matter how widespread.

Systemic Lupus Erythematosus with Nodular Lesions.—Dr. W. Frain-Bell (for Dr. L. Forman).

Mrs. O. L—, aged 28.

History.—In February 1954 erythema and oedema appeared on the cheeks, chin, forehead, nose and ears. A few weeks later small nodules were noticed on the face and later on the V of the neck and on the arms. Chronic discoid lupus erythematosus

was diagnosed and mepacrine 300 mg. daily was given for six weeks without benefit. Lesions were fading after August 1954, but fresh nodules appeared in January 1955 and have persisted. More recently muscle pains and joint stiffness have been noticed.

Previous history.—Plastic peritonitis at age 14. Synovitis of right foot in 1953.

On examination.—There was extensive involvement of the skin of the face with lupus erythematosus, together with some nodular lesions on the forehead. On the upper limbs there were erythematous papules and scaly pigmented plaques, while on the trunk there were numerous small skin-coloured nodules showing no redness or scaling. Enlarged glands were present in both axillae and groins and in the left supraclavicular region. A systolic murmur present at the apex, and an intermittent pyrexia.

Histology (Dr. Haber).—Cheek : There is hyperkeratosis and follicular plugging with oedema of the basal cell layer. The corium is oedematous and exhibits a round cell infiltration within the cutis, partly perivascular and partly periacinous.

Nodule from arm : the epidermis does not show any diagnostic features. There is much inflammatory reaction and cobweb-like degeneration of the collagen.

Investigations : (1) Haemoglobin 74% (11·3 g.) ; RBC 3,880,000 ; WBC 4,400 ; polymorphs 69%, lymphocytes 24%, monocytes 5%, eosinophils 2% ; ESR 34 mm. in 1 hr. (2) Typical L.E. cells present in considerable numbers in the peripheral blood. (3) Serum proteins : Total 6·7 g.% ; albumin 3·4 g.% ; globulin 3·3 g.% ; (4) W.R. and Kahn negative. (5) No cold agglutinins in any dilution. (6) Urine, scanty RBC, WBC and granular casts. (7) Mantoux : negative 1/10,000.

Comment.—This unusual nodular eruption in association with systemic lupus erythematosus was first described by Gold (1954) and more recently by Silva and Portugal (1955). In Gold's patient the nodular eruption appeared to have been the first feature of the illness and preceded the erythematous eruption by some weeks. It also seemed to have fluctuated widely from week to week, sometimes being scarcely visible.

In these cases the most striking histological change in the dermal nodules was a peculiar cobweb degeneration in the connective tissue and in the case presented this change was present although mucin was not demonstrable.

References.

Gold, S. C. (1954) *Brit. J. Derm.*, **66**, 429.
Silva, R. E. and Portugal, H. (1955) *Ann. Derm. Syph., Paris*, **82**, 34.

Dr. H. Haber : A conspicuous feature of the histology is the finding of a network of fine mucin-like threads filling out the tissue spaces of the collagen. They do not appear to be a deposit but rather a peculiar type of collagenous degeneration which I like to call "cobweb"-like. It would be, however, erroneous to regard these changes as peculiar to subacute lupus erythematosus, as they are also found in dermatomyositis, in reticuloses, non-specific inflammatory reactions and in the vicinity of malignant tumours. The nature of this type of degeneration is being studied.

Dr. J. E. M. Wigley : Had the patient had any mepacrine before the lesions appeared ? It does raise the question of lichenoid eruption.

Dr. Frain-Bell : The small nodular eruption was already present in some areas before the course of mepacrine was started.

Naevo-xantho-endothelioma.—Dr. M. Feiwel.

S. L—, an infant girl aged 5 months.

A tumour on the scalp was noticed when she was three weeks old ; it gradually grew in size and became covered with a scab with some tendency to ulceration and discharge. Other nodules appeared on the scalp and trunk, and are still arising. Two were excised, one from the scalp and one from the back.

The histological report (Dr. P. L. Masters) was :

" Two subcutaneous nodules, 8 and 10 mm. in diameter, uniformly pale yellow on section. Histologically they consist of closely-packed cells with large round or

kidney-shaped vesicular nuclei and a moderate amount of variably foamy cytoplasm. Many of the cells are multinucleated and there is a considerable number of Touton type giant cells. The deeper part of the larger nodule contains many eosinophils and has involved the subcutaneous fat.

" Diagnosis : Histiocytoma of the naevo-xantho-endothelioma type."

On examination.—Smooth, soft, shiny, yellow dome-shaped nodules with a reddish tinge are present, three on the scalp and one on the back of the right shoulder of about 1 cm. diameter. Below the one on the back are two smaller lesions which have only lately arisen.

Comment.—This disorder has not yet been satisfactorily classified. Lever (1954) withdraws it from the xanthoma group and regards it as a histiocytoma that differs from the usual histological picture only by showing foam cells and giant cells of the foreign body type in large numbers. Tannhauser (1950), however, equates naevo-xantho-endothelioma with juvenile (infantile) xanthoma and considers it to be the monosymptomatic variety of the eosinophilic xanthomatous granuloma occurring in infants, involving the skin only and usually carrying a good prognosis. Andrews (1954) divides juvenile xanthoma into two groups, the infantile xanthoma which is again the monosymptomatic form of the eosinophilic xanthomatous granuloma, and a second group which is naevo-xantho-endothelioma. Forman (1951) presented a baby patient to this Society with the diagnosis of juvenile xanthoma and discussed McDonagh's (1912) concept of the naevo-xantho-endothelioma ; other members considered his case belonged to the latter group. Ormsby and Montgomery (1954) in their last edition say that they no longer distinguish juvenile xanthoma from naevo-xantho-endothelioma. Calnan (1952) reviewed cases from the Hospital for Sick Children, Great Ormond Street, under the title " Juvenile Xanthoma (McDonagh)."

References.

Andrews, J. C. (1954) *Diseases of the Skin*, 4th edition. Philadelphia : W. B. Saunders.
Calnan, C. D. (1952) *Great Ormond Street J.*, **4**, 127.
Forman, L. (1951) *Brit. J. Derm.*, **63**, 74.
Lever, W. F. (1954) *Histopathology of the Skin*, 2nd edition. London : J. B. Lippincott Co.
McDonagh, J. E. R. (1912) *Brit. J. Derm.*, **24**, 85.
Ormsby, O. S. and Montgomery, H. (1954) *Diseases of the Skin*, 8th edition. London : Henry Kimpton.
Tannhauser, S. J. (1950) *Lipidoses*, 2nd edition. New York : Oxford University Press.

The following cases have been published in *Proc. R. Soc. Med.*, **48**, 772 :

Turban Tumours of Scalp, with Bony Erosion (Cylindroma).—Dr. J. B. Lyon.

Colloid Milium.—Dr. H. T. H. Wilson.

The following cases also were shown :

Lichen Sclerosus et Atrophicus.—Dr. E. J. Moynahan.

Naevus of Ota.—Dr. H. Haber and Dr. M. Feiwel.

Hypertrophic Lichen Planus Treated with Local Injections of Hydrocortisone.—Dr. J. S. Pegum.

Acneiform Papulonecrotic Tuberculide.—Dr. R. H. Meara.

A case of **Torulosis** was reported by Dr. I. S. Hodgson-Jones.

NORTH BRITISH DERMATOLOGICAL SOCIETY.

Glasgow, 13 October 1955.

Epidermolysis Bullosa Simplex with Hyperextensibility of Joints.—Dr. W. A. Dewar.

A girl aged 5.

History.—Bullae were noted on hands and feet within a few days of birth. Since then recurrent bullous lesions have appeared on the soles, fingers, vulva and face and, in general, have been more frequent during summer months.

Family history.—No parental consanguinity. Paternal grandmother had "alkali dermatitis". No skin disorder present in maternal forebears as far back as great-grandparents.

Examination.—Bullae present on soles, fingers, wrists, vulva and right cheek. Some lesions on hands are haemorrhagic. On the vulva and the face the lesions are grouped and some show crescentic configuration. No scarring, atrophy or milia formation is present. Hyperextensibility of elbow and knee joints is evident.

Investigations.—Detailed examination of peripheral blood showed no haematological abnormality, but there was a positive Hess's test. Examination of urine revealed no indican and no abnormal excretion of porphyrins or their precursors. Potassium iodide (50% in paraffin) patch test negative after 24 hours. Ophthalmoscopy revealed no abnormality. W. R. negative. Blood chemistry normal.

Histology (Dr. J. A. Milne)—A vesicle is present in the epidermis. There is no elastic dystrophy.

Treatment.—Sulphapyridine 0·5 g. daily for 14 days resulted in no improvement. Attempts to prevent bulla formation by pressure bandaging have been only moderately successful.

Comment.—The case presents features of the Weber-Cockayne type of epidermolysis bullosa together with joint signs suggestive of Ehlers-Danlos syndrome. A superficial resemblance to juvenile dermatitis herpetiformis is evident, as in Dowling and Meara's (1954) cases. This may result from trauma occurring around the mouth and vulva, and trauma is certainly a factor in the occurrence of bullae on the hands and feet.

Everall (1954) and also Caldwell (1955) have demonstrated co-existing tylosis in such cases and the present case is a further illustration of mixed congenital defects. The positive Hess's test may indicate that the manifestations of this disease result from a primary defect of vascular mechanisms as suggested by Winer and Orman (1945).

References.

Caldwell, I. (1955) *Brit. J. Derm.*, **67**, 315.
Dowling, G. B. and Meara, R. H. (1954) *Ibid.*, **66**, 139.
Everall, J. (1954) *Ibid.*, **66**, 54.
Winer, M. N. and Orman, J. M. (1945) *Arch. Derm. Syph., Chicago*, **52**, 317.

Distinctive Exudative Discoid and Lichenoid Chronic Dermatosis of Sulzberger and Garbe.—Dr. A. Girdwood Fergusson.

A man aged 64, of Scottish ancestry.

History.—He has suffered from a recurring, non-irritating, generalized skin eruption over the past 15 years. Prior to my seeing him the condition had proved resistant to all types of topical applications, but localized areas had shown some temporary healing after fractional x-ray therapy in small dosage.

Past history.—He had always been facile in character.

Examination (23.x.53).—Sharply demarcated oval and discoid plaques, sometimes moist and crusted and at others of psoriasiform or lichenoid character, are present on the skin of the forehead, face, trunk (particularly mammary regions and upper part of the back) and limbs (Fig. 1). The lesions show varying degrees of infiltration. Marked erythema and desquamation are present on the skin of the 4th–5th toe interspaces of both feet. There is no lymphadenopathy and the liver and spleen are not palpable.

Fig. 1.

Investigations.—B.P. 170/95.

Hb 110%, PCV 45%, RBC 5,400,000. WBC 8,500 (neutros. 47%, lymphos. 44·5, monos. 5, eos. 3, basos. 0·5%).

Serum proteins 7·5 ; alb. 4·8, glob. 2·7.

W. R. negative.

Blood sugar curve within normal limits.

ECG : left ventricular preponderance.

Fractional test meal normal.

Urine.—Sp. Gr. 1024. Occasional green or orange reduction with Benedict's reagent (renal glycosuria).

Abundant mycelium (*E. floccosum*) was demonstrated from the affected toe interspaces.

Psychiatrist's report.—Congenitally subaverage intelligence.

Histology (Dr. J. A. Milne).—2.xi.53 : Sections show a subacute eczematous process. In the dermal infiltrate plasma cells are numerous, and this raises the possibility of Sulzberger and Garbe's syndrome.

3.ii.54: No significant change. No evidence of mycosis fungoides. 4.iii.55: Superficial ulceration of the epidermis with serous crusting. There is dense inflammatory infiltrate in the upper cutis along the dermal appendages. The predominant cell, as in previous biopsies, is the plasma cell and the general picture is similar to that of previous sections though the quantity of infiltrate is much greater.

Treatment and progress.—Treatment of the focus of fungus infection on the feet (prior to steroid therapy) resulted in its clearance but in no improvement as far as the generalized eruption was concerned. A marked improvement took place when aqueous solution of ACTH was administered intramuscularly in dosage of 100 mg. daily, but relapse occurred on reduction to 50 mg. daily. A renewed response was observed with 40 mg. of ACTH gel twice daily, but active lesions persisted and the treatment was gradually discontinued only to be resumed following an unsuccessful trial of general ultraviolet light baths.

Dosage has ranged between 150 and 15 mg. daily (ACTH solution) depending upon response, and ACTH has been from time to time combined with cortisone, from 250 to 50 mg. daily by mouth. A recent controlling dose has been ACTH gel 20 mg. and oral cortisone 50 mg. daily.

Comment.—Points of interest in this case are the marked clinical resemblance to mycosis fungoides, the entire absence of itching and the ancestry of the patient. Further, it is notable that steroid therapy appears to be necessary if the disease process is to be kept under even reasonable control.

Reference.

Sulzberger, M. B. and Garbe, W. (1937) *Arch. Derm. Syph., Chicago*, **36**, 247.

The following cases also were shown:

Epidermolysis Bullosa Dystrophica (3); Chronic Arsenical Poisoning; Hard Naevus of Brocq; Favus.—Dr. W. A. Dewar.

Necrobiosis Lipoidica; Pellagra.—Dr. Christina C. McEwan (for Dr. A. Girdwood Fergusson).

Pyoderma Gangrenosum; Pityriasis Rubra Pilaris; Pemphigus Foliaceus (3); Leprosy; Mycosis Fungoides.—Dr. J. C. P. Logan (for Dr. A. Girdwood Fergusson).

BOOK REVIEWS.

A Compendium of Ethical Proprietaries.*

This little book consists mainly of an alphabetical list of proprietary drugs giving their composition, indications, dosage and "pack". It is cross-indexed with a list of drugs, indicating in what proprietary preparations they are to be found.

There is also a therapeutic index—an alphabetical list of diseases and symptoms with the drugs recommended for them. It is not so easy to understand for whom this list is intended, including as it does, "Skin Blemishes," the only treatment for which is Rosul; Rosul is described as a masking cream containing ½% of sulphur, "useful in covering lupus erythematosus and other blemishes."

A further index gives the names and addresses of drug manufacturing firms.

Within its limits the book certainly has its value, chiefly in that it enables quick identification of proprietary remedies; it will also greatly facilitate the exact recognition of drug allergies when the responsible substance is wrapped up in a proprietary ointment or pill. At other times it may help in the choice of suitable ways of prescribing a desired treatment. F. R. B.

Lesions of the Mouth.†

This is a beautifully produced book with many black and white photographs and photomicrographs; these are outstandingly good except for one or two of the depth of the oral cavity. The author has in mind not only dermatologists but others, including dental surgeons, interested in this region. Treatment is only sporadically touched upon and in the reviewer's opinion could well have been omitted. M. S. S.

Colour in Dermatology.‡

Show a class of medical students a typical case of lichen planus, ask them the colour of the lesions, and you will receive an astonishing variety of replies. From this it can be inferred that the colour of objects depends on the eye of the beholder and, therefore, description by colour is only a very rough business.

Each one of us looking at a patch of erythema probably observes something slightly or greatly different. It is, therefore, quite possible that in dermatological teaching colour could be disregarded altogether. Indeed it seems likely that a colour-blind person would usually make an accurate diagnosis of most skin diseases for he has to rely alone on the shape, distribution, form, grouping of the lesions and so on, together with symptoms such as itching, pain and burning.

When we come to coloured photographs we are up against something quite different, for the camera, or more particularly colour film material, does not see in the sense that

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* "*The Compend*": *A Compendium of Ethical Proprietaries used in Medicine and Pharmacy.* W. Hetherington, F.P.S. (1955) Bristol: John Wright & Sons, Ltd. Pp. 704. Price 32*s.* 6*d.*

† *Dermatology of the Oral Cavity and of the Circumoral Region.* Alloys Greither. (1955) Stuttgart: Georg Thieme. Pp. 262. Illus. 191. Price DM 39.60.

‡ *An Atlas of Regional Dermatology.* G. H. Percival and T. C. Dodds. (1955) London: E. & S. Livingston Ltd. Pp. 264. Illus. 475. Price £5.

we understand it. It is a chemical process governed by the illumination, the quality of the lens used, filtration and, above all, processing. It is altogether too rigid and may even be misleading by giving too uniform a quality in its reproduction of colour. It is unfortunate for dermatology that colour film behaves at its most chancy and wildest when it comes to the range of reds and in the excellent work under review this is particularly borne out.

Colour printing is still at a very rudimentary stage and it is very much a matter of hit or miss. Some of the illustrations in this work are excellent. In others the colour values, particularly the reds, are poor and some of the plates are fuzzy. This fuzziness, I imagine, may vary from copy to copy of the book.

Having made this criticism, one must state that a set of illustrations such as this showing the differential diagnosis of skin lesions by regions is most valuable and instructive; but one is bound to admit that had the work been produced in monochrome the illustrations would have been just as useful and, in the case of some of the illustrations where the colours are misleading, very much better. This would have enabled the work to be produced at far lower cost. J. L. F.

CURRENT LITERATURE

CORTICOTROPHIN AND CORTISONE IN THE TREATMENT OF SCLERODERMA. MM. Zion, B. Goldberg and Suzman. (1955) *Quart. J. Med.* n.s., **24**, 215.

Fourteen patients with various types and stages of scleroderma were treated with cortisone or corticotrophin or a combination of these hormones. In one patient there was complete remission, in four, moderate to much improvement and in nine slight improvement. Best results were obtained in patients with no peripheral vascular disturbances and in those where the duration of the disease was less than six months. In none was benefit maintained when treatment was temporarily discontinued. J. M. B.

MEPACRINE THERAPY IN CHRONIC LUPUS ERYTHEMATOSUS. E. Thiel. (1955) *Derm. Wschr.*, **132**, 889.

Fifty-five cases of chronic lupus erythematosus were treated with mepacrine. All received 300 mgm. daily until the skin became quite yellow and after that 100 mgm. daily for weeks.

Forty-three cases showed satisfactory therapeutic effects, 12 showed side-effects, all of which were reversible. Some of the cases showing intolerance belonged to the group of cases treated successfully.

Side-effects were gastroenteritis, hepatitis and headaches, vomiting and nausea. W. G. T.

MULTIPLE CUTANEOUS GANGRENE IN VASCULAR DISEASE. G. Weber. (1955) *Derm. Wschr.*, **132**, 913.

A number of cases of cutaneous gangrene are undoubtedly due to vascular disease. The clinical appearances do not allow any conclusion as to the vascular processes underlying the gangrene. One and the same vascular process can cause different cutaneous appearances.

Two cases of multiple cutaneous gangrene observed by the author are reported in detail. Biopsy sections from the gangrenous areas did not allow a definite diagnosis in either case. Generalized arteriosclerosis was established at autopsy as the cause in one case, in the other examination of an amputated leg proved gangrene to have been due to endarteritis obliterans. It is of interest that the gangrene which had been dry hitherto, became moist in both cases when thrombosis of the abdominal aorta occurred. W. G. T.

HYPOPROTHROMBINAEMIA AND THEORETICAL BASIS FOR LIVER THERAPY IN HERPES ZOSTER. St. Pastinszky, E. Kovács, St. Rácz and O. Geszti. (1955) *Derm. Wschr.*, **132**, 937.

The authors found marked hypoprothrombinaemia in all of 21 cases of herpes zoster. Symptoms and hypoprothrombinaemia remained unaffected by administration of vitamin K, but all of 17 cases showed return to normal of the prothrombin level and disappearance of pain within 24 hours after liver injections. W. G. T.

THE ANTIMYCOTIC ACTION OF THE ESTERS OF p-OXYBENZOIC ACID. J. Uri, R. Bognár, St. Békési and M. Balogh. (1955) *Derm. Wschr.*, **132**, 942.

In vitro tests with various esters of p-oxybenzoic acid were carried out. It was found that the esters of higher alcohols had a stronger fungistatic action when used on cultures of trichophyton, epidermophyton, microsporon and achorion. They were also less toxic in animal experiments. W. G. T.

HYDROCORTISONE OINTMENT IN THE TREATMENT OF CHILDREN WITH ATOPIC DERMATITIS. B. J. Heilesen, A. A. Kristjansen and E. L. Reymann. (1955) *Acta allergol.*, **8**, 314.

Thirty-nine children with atopic dermatitis were given prolonged treatment with a 1% hydrocortisone ointment. The results were good in 23, doubtful in 9, and poor in 7 cases. H. T. H. W.